Developmental Dental Defects and Tooth Wear: Pathological Processes Relationship

Chapter

Developmental Dental Defects and Tooth Wear: Pathological Processes Relationship

Francesco Grande and Santo Catapano

Abstract

Many conditions or pathologies can modify teeth surfaces and cause several functional and esthetic problems. Congenital dental defects and tooth wear are two of the most important reasons of dental tissue changes. Nowadays, the prevalence of tooth wear is increasing because of a high incidence of non-physiological tooth wear especially in young people. However, distinguishing dental defects originated from tooth wear or developmental dental defects is crucial to plan the most suitable treatment. Then the aim of this work is to present the different pathological conditions caused by these two etiological factors as well as the underlying biochemical mechanisms and incorrect habits related.

Keywords: tooth wear, amelogenesis imperfecta, dentinogenesis imperfecta, attrition, abrasion, erosion, abfraction

1. Introduction

Many conditions or pathologies can modify teeth surfaces and cause several functional and esthetic problems to the dental patient. They could be divided in:

- congenital defects or developmental dental defects;
- acquired dental defects.

Congenital dental defects include pathologies as amelogenesis imperfecta, dentinogenesis imperfecta and molar-incisor hypomineralization.

On the other side, dental caries, occlusal trauma and tooth wear are recognized as the most important reasons of dental tissue changes, concerning acquired dental defects. However, tooth wear has always been underrated and less considered than dental caries and trauma [1, 2]. Also congenital dental defects are little considered because of the lower prevalence in the population then dental caries although there is a clear association between some types of developmental defects and dental caries in primary dentition [3].

Regarding tooth wear, today the common opinion of dental clinicians is that the prevalence of tooth wear is increasing, because of a high incidence of non-physiological tooth wear and this is confirmed by important surveys [4, 5]. Also the prevalence of extensive wear is thought to increase, especially erosive tooth wear at young age [6].

Regarding congenital dental defects, the comprehension of genetic and environmental influences on enamel and dentine development are considered crucial for preventive actions and treatment planning of these conditions [7].

With the increased life expectancy and augmented frequency of oral hygiene procedures, problems related with tooth wear and congenital teeth defects are likely to place greater demands upon dental clinicians.

Then, in order to face that, it is important to understand the pathological mechanisms underlying developmental dental defects and dental wear and what biochemical processes and incorrect habits are involved in these conditions.

2. Congenital dental defects

Congenital dental defects are due to inherited or spontaneous genetic or epigenetic mutations that influence specialized cellular and biochemical pathways involved in dental hard tissue formation [8]. Local or systemic defects depend on where affected genes are expressed [9, 10].

However, these conditions are also caused by environmental factors such as drugs, infections, nutritional deficiencies, medical conditions or trauma [7]. Clinical importance of these defects is related to the risk of tooth decay, especially in respect of biofilm retention [11]. In addition, problems in restorative treatment because of the effectiveness of the materials and cements used for patient rehabilitation could be present.

2.1 Developmental enamel defects

Developmental enamel defects are mostly due to mutations in genes that code for enamel proteins. Generalized systemic conditions may also be present and could involve neuroectodermal mesenchyme tissues, that share common embryologic origins with enamel and dentin [12]. Otherwise, they could be induced by some pre-, peri- and postnatal factors.

Clinically, enamel abnormalities due to gene mutations are grouped under the name of amelogenesis imperfecta (AI) [7] and can be clinically divided into qualitative and quantitative defects. Qualitative defects differ from quantitative ones because they are characterized by the presence of normal amounts of enamel that is deficiently mineralized while quantitative defects are referred to enamel quantity.

Hypoplasia is a quantitative reduction of enamel formation due to disruption in ameloblast production. It can affect both the primary and permanent teeth [13]. The etiology of hypoplasia is related to insults occurring during the earliest stages of enamel development (matrix formation) [14]. It causes pits, grooves, thin or missing enamel, dental surface breaks and deficiencies.

Hypomineralization is a qualitative defect due to insults occurring in the calcification process. The resulting reduced mineralization could be recognized as soft enamel. When an altered translucency or opacity affects the entire tooth, or a localized area we can also talk of hypomaturation [12]. In case of hypomaturation and/or hypomineralization, enamel could fracture easily under loading [15] and this could result in severe tooth wear.

In the field of hypomineralization defects, a peculiar type of chronological enamel hypomineralization is the molar-incisor hypomineralization (MIH). It determines well demarcated opaque areas on the surface of permanent molars and incisors that could be colored from white to yellow or brownish, depending on the severity of the pathology [16]. In these cases, teeth often show enamel disintegration at the occlusal surfaces, post eruptive tooth structure loss and high

caries susceptibility. Tooth sensitivity could also be present because of the porous prismatic enamel morphology [17]. This condition may predispose to tooth wear due to attrition between teeth and it can be aggravated in presence of other factors as abrasion and erosion. The severity of the clinical status may require extensive treatment [18–20].

Systemic factors affecting enamel development may also be distinguished in pre-, peri- and postnatal conditions in relation to the timing of the event [18] and could be caused by metabolic disturbances, drugs consumption, local infections, trauma and radiation [21, 22].

Amelogenesis defects may predispose to tooth sensitivity, plaque accumulation and increased caries risk, and in severe cases even space loss and malocclusion [23]. Also tooth wear can be associated to developmental enamel defects [7]. Infact, tooth wear could be a detrimental consequence of attrition between teeth in case of amelogenesis imperfecta and this may also cause the alteration of the normal occlusal pattern. Qualitative enamel defects may decrease the resistance of teeth both to erosion and abrasion because of the weak resistance to acid attacks and friction with foreign bodies. Furthermore, the augmented risk of dental caries and the porosity of enamel structure can enhance the process of tooth breakdown due to occlusal loading. Anterior open bite and increased calculus formation are commonly encountered in association to amelogenesis imperfecta [15] and could worsen the oral condition. Also tooth wear can be associated to developmental enamel defects.

2.2 Developmental dentin defects

Developmental dentin defects principally origin from mutations in genes coding for the proteins involved in type 1 collagen or in the extracellular matrix as well as in the mineralization processes. Defects may involve only dentine or both dentine and skeleton, if altered proteins are specific to dentine or expressed both in bone and dentine. These two types of clinical phenotype classified inherited dentine defects in the Shield's classification system [24].

Dentinogenesis imperfecta is the most common type of developmental disorder of dentine, affecting both primary and permanent teeth. It is sometimes associated with osteogenesis imperfecta [25]. When dentine and osseous defects are associated, there is a genetic fragile bone condition together with a reduced support of dentine that could show an opalescent brown discoloration. Lacking teeth support leads to easily fractures of the overlying enamel fractures as well as rapid wear and attrition of the teeth. Progressive pulp obliteration usually begins soon after eruption of the teeth and wear could arrive to the gingival level [7]. Dentine dysplasia is less common and shows normal appearing crowns with normal or short roots and pulp reduced in size. Occasionally, other abnormalities such as dental discolorations, bulbous crowns and pulp obliterations may be encountered [26].

Dentin developmental defects are highly expressed in familiar hypophosphatemia, also known as 'vitamin D-resistant rickets', an X-linked dominant inheritance condition [27]. This condition is associated with reduced resorption of phosphate in the renal tubules and characteristic rachitic bone deformities [28]. Spontaneous dental abscesses in children with no history of caries or trauma showing teeth involved in familial hypophosphatemia may occur [29]. Poorly mineralized dentine, and tubular defects extended closed to the dentino-enamel junction could predispose the pulp to exposures and infection as soon as the enamel is removed (superficial caries or attrition) [28, 30].

Because of the X-linked condition, boys are affected by the most severe dental involvement and girls the least. A wide range of spectrum manifestations has been described [26].

3. Tooth wear mechanisms

Tooth wear is defined as the progressive loss dental hard tissues from the surfaces of the teeth, caused by relative motion (friction) at the surface [1]. This type of wear includes attrition and abrasion, but also dental erosion and abfraction are nowadays included in this condition.

Tooth wear due to masticatory function is regarded as a natural phenomenon and a certain degree of tooth wear is considered unavoidable during age [31]. If the degree of destruction or the rate of loss becomes excessive, overcoming the physiological mechanisms of compensation (e.g. formation of secondary dentin), problems arise with the necessity of treatment [32]. It may cause functional and esthetic problems, dental sensitivity [1], or it could prejudice the survival of the teeth [2]. Wear could be critically pathological when it leads to poor masticatory function with concomitant reduction in quality of life and possible deterioration of systemic health [33].

The presence of developmental dental defects of enamel or dentine origin could enhance the process of tooth wear. In fact, decreased resistance in teeth with enamel and dentin abnormalities is a fact and the etiological mechanical and chemical processes of attrition, abrasion, erosion and abfraction may critically reduce the survival rate of teeth with developmental dental defects.

Then, understanding and recognizing the disruptive processes of tooth wear and if it hides possible developmental dental defects is necessary to prevent and treat several dental pathologies as worn dentition.

3.1 Attrition

Attritional is defined as the loss of tooth tissue due to friction between opposing teeth and is thus related to dental occlusion. The progressive tooth substance loss (TSL) is considered by Berry and Poole [31] a normal aging process, in which formation of secondary dentine, muscle adaptation, alveolar growth and attrition are all part of a compensation mechanism. In this view, attrition, as a normal process of changing dental morphology, should not be regarded as excessive. However, the loss of tooth tissue usually affects the dental occlusion, and it is still controversial the fact of ignoring a changing occlusion in the management of dental problems such as 'extensive' attrition or temporomandibular disorders. For these reasons and because of different assessment criteria, the exact prevalence of attrition is unclear [1, 2].

The literature on attrition does not provide clear evidence for the efficacy of particular occlusal designs in the management of attrition [34, 35]. Some cross-sectional studies [36, 37] indicate that anterior (spatial) relationships and attrition were related. As expected, anterior guidance, which is partially determined by vertical overbite and horizontal overjet, seems to reduce the risk for posterior attrition, but increases the risk for anterior attrition. Canine protection, that ensures anterior guidance, may reduce the posterior tooth substance loss but only one study tried to demonstrate it [37]. Absent posterior support did not necessarily lead to increased attrition of the remaining teeth, whereas a reduced number of teeth may lead to increased wear of the remaining teeth [38].

In dentinogenesis imperfecta, attrition is deleterious. As reported in literature, the reduced support of dentine due to genetic condition leads to easily fractures of the overlying enamel and to a progressively rapid tooth wear caused by attrition [7].

For this reason, attrition is very common in dentinogenesis imperfecta and have to be considered as one of the most important factors of tooth wear.

Attrition in patients with amelogenesis imperfecta may result in widespread exposed dentin both in primary and in permanent teeth. Deficiencies in enamel attachment to dentin and defective enamel structure take part in the process of

tooth wear, that could be faster and result in dentoalveolar abnormalities because of the continuous eruption of teeth [39].

In Molar-incisor hypomineralization (MIH), tooth substance loss could be enhanced due to attrition mechanisms [40]. MIH complicated with tooth substance loss may not only compromise the esthetics and function but also endanger the pulp and longevity of the affected teeth. Tooth substance loss might be complicated by eruption of the teeth with its dentoalveolar processes which obliterate the space for any restorations [41].

Attention in these patients should also be placed when a prosthetic restoration is performed on the antagonist tooth because of the possible increased wear. The material choice is fundamental regarding mechanical properties, hardness and patient occlusion scheme, and the prosthetic restoration of the antagonist with the same material could be considered.

Attrition may be accelerated by "demastication", intended as a tooth wear process occurring during mastication of food influenced by the abrasiveness of the individual food particles [42, 43]. High levels of inorganic compounds and salts were found in snuff by Dahl et al. [44] while silica abrasive particles were also discovered in tobacco chewing by Bowles et al. [45].

Despite the possible augmented tooth substance loss because of the food particles abrasiveness, a restorative rehabilitation of the patient with developmental dental defects is important also for reestablish an appropriate food intake. In fact, the tooth wear and pain disturbances evoked by some types of food may altered patient's alimentary habits, avoiding the consumption of some important nutrients [46, 47].

Some parafunctional habits (bruxism and clenching) may contribute to attrition [1, 48]. One study concluded that excessive forceful grinding during ongoing sleep bruxism events may cause canine attrition (**Figure 1**) [49]. While the prevalence of bruxism is unclear, studies report between 5–96% of the population may be affected [1]. Its prevalence on population with developmental dental defects is not reported in literature but considering the weakness of the tooth tissues in these patients, it could be responsible of a severely worn dentition in young age [50]. Night bruxism and clenching are detrimental and a thorough dental and muscular examination has to be carried out to identify signs of bruxism and clenching in order to avoid major dental destruction. A misdiagnosis may involve future complex oral rehabilitations in order to treat patients with developmental dental defects and severe worn dentition [50].

3.2 Dental abrasion

Dental abrasion is defined as the loss of tooth substance due to friction with food and foreign body (e.g. toothbrush) and may obliterate attrition wear patterns

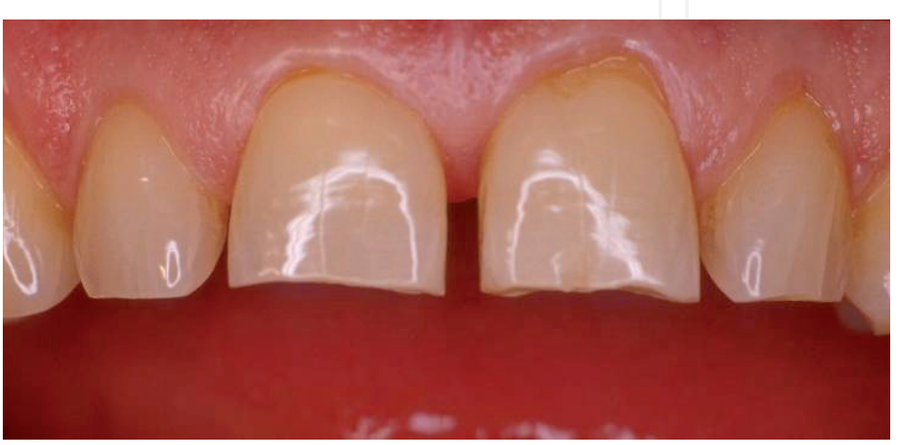

Figure 1.
A case of teeth attrition caused by bruxism.

caused by friction of opposing teeth [51]. Some types of dental abrasion may be related to habit or occupation [1, 2, 52]. Notching of incisal edges may be caused by pipe smoking, nut and seed cracking, nail biting, and hairpin biting [51, 53].

The etiology may also be deduced from the location and pattern of abrasion [52, 54]. The most common cause of dental abrasion can be found at the cervical areas and is related to toothbrushing. The technique applied, the time and frequency, the bristle design, and also the dentifrice used during toothbrushing can strongly influence this pattern [1, 48, 52]. A zealous, vigorous and repeated toothbrushing as well the use of toothbrush with not rounded tips of the bristles and abrasive dentifrice, could lead to an important dental abrasion.

In literature studies, premolars were more frequently affected with lesions varying from wedge-shaped and dish-shaped to flattened irregular and concave, with several depth and size (**Figures 2** and **3**) [55]. Data analysis revealed that vigorous toothbrushing is the major etiologic factor [56–60].

In patients with developmental dental defects, it is important to place a strong emphasis on an adequate oral home-care regimen. Education of the patient and parent guardian on an adequate tooth brushing technique and recommended oral

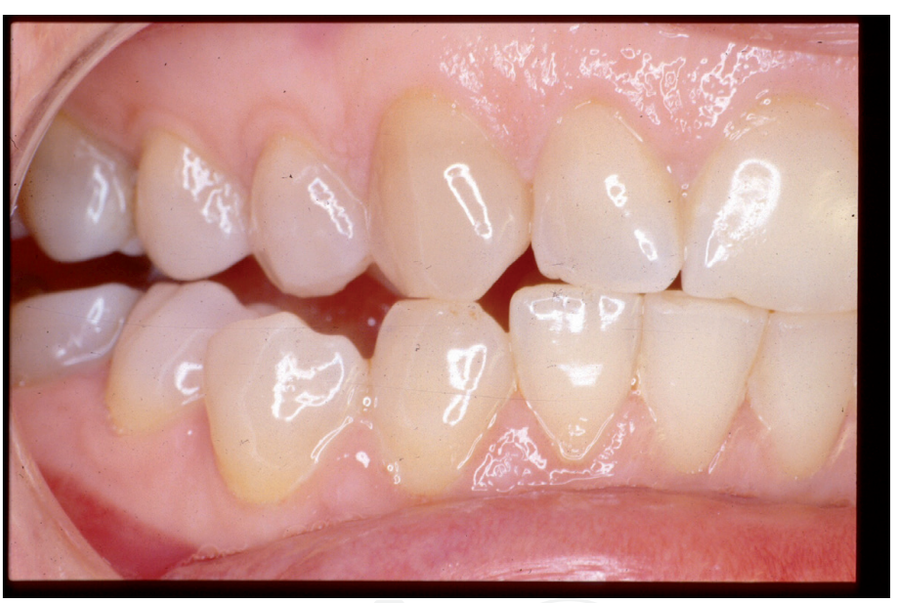

Figure 2.
Mild abrasion in canine and premolar teeth.

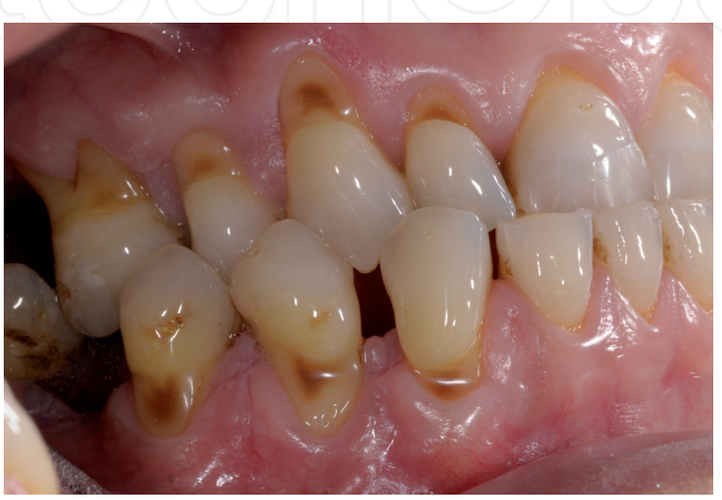

Figure 3.
Severe abrasion and abfraction lesions of the first and fourth quadrant teeth.

hygiene habits is required. Pitted enamel surfaces and roughness of teeth especially in amelogenesis imperfecta may predispose plaque accumulation and augmented susceptibility to dental caries. Oral hygiene could be poor in some patients, often because of tooth hypersensitivity and the presence of an anterior open bite associated with mouth breathing [61]. Patients have to be informed regarding their situation and instruct to maintain correct oral hygiene habitudes.

Motivation to home oral hygiene instructions is important not only for the health of hard dental tissues but also for the soft gingival tissues. Restorative procedures are usually performed in patients suffering of enamel and dentine defects. Then, teeth surfaces may retain more plaque and gingival hyperplasia can be expressed near restorations.

Dental abrasion is principally associated with horizontal brushing technique [56], but also with brush stiffness [62, 63], dentifrice abrasiveness [57, 58] and age [64, 65]. It was observed that hard bristles caused the least amount of tooth abrasion while soft bristles caused the most amount of abrasion, because of the major retention of toothpaste offered by smaller diameter filaments and denser tufts [66, 67].

Although studies show a strong association of cervical abrasions to toothbrushing, some authors affirm that dental erosion plays a great role in this tooth wear [1, 48, 63]. Experiments show that an interval of 1-hour should be considered before toothbrushing after an acid attack, in order to allow a period of remineralization necessary for improving the resistance of eroded enamel against brushing abrasion [68, 69]. Seong et al. [70] observed that enamel repair commences within 2 hours following an acidic attack and is completed 4–24 hours later. Then it could be concluded that the enamel repair process is relatively slow, exposing to high risk of tooth wear mediated by erosion/abrasion. In this context, patients with developmental dental defects and especially with enamel hypomineralization should have particular attention to avoid toothbrushing after eating acid foods and drinks. In this context, two mechanisms could accelerate tooth wear: the increased demineralization after an acid attack due to the enamel matrix hypomineralization and the reduced rate of remineralization caused by the alteration of the enamel matrix [14].

Obviously, the amount of saliva produced by each patient is one of the most important protective factors to avoid erosion of tooth structure. An appropriate evaluation of salivary rate production should be performed in this sense.

Dental hypersensitivity related to cervical abrasion and exposure of dentin to the oral environment may be possible and generally more frequent than in other populations [71].

3.3 Dental erosion

Erosion is known as the dissolution of the surface of an object by means of fluids. Dental erosion is always caused by acid dissolving hard tooth tissues [72] and has been defined as the irreversible loss of dental hard tissue caused by a chemical process not involving bacteria (**Figure 4**) [43].

A general trend of increasing tooth wear by acid erosion in particular, amongst the young people, was highlighted by several authors [73–75]. In particular, young women (15–25 years old) are often affected by psychosomatic eating disorders [76].

These phenomena often clinically overlap with other clinical pathologies such as abrasion and attrition (**Figure 5**). This could lead to a difficult differentiation, especially at the initial stages. However, as the degree of erosion increases, a more suitable differential diagnosis can be performed. It is very important to establish if dental erosion underlines any developmental dental defect that may contribute to the pathologic condition observed. And it is already fundamental to understand what type of developmental defects may affect the dentition similarly or in addition

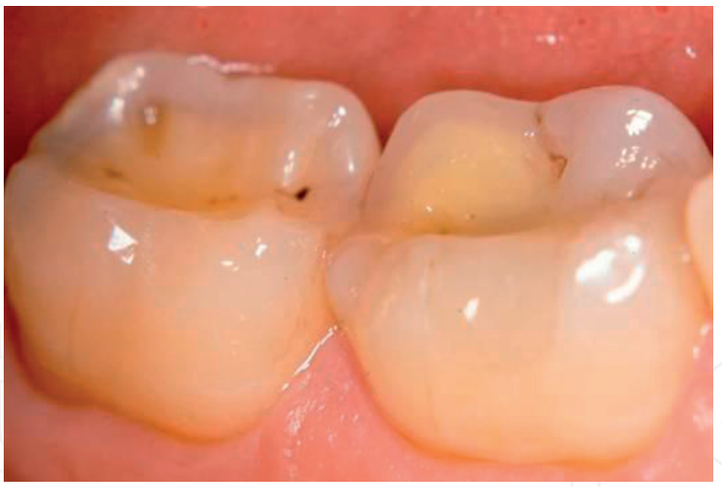

Figure 4.
Occlusal erosion of molar teeth.

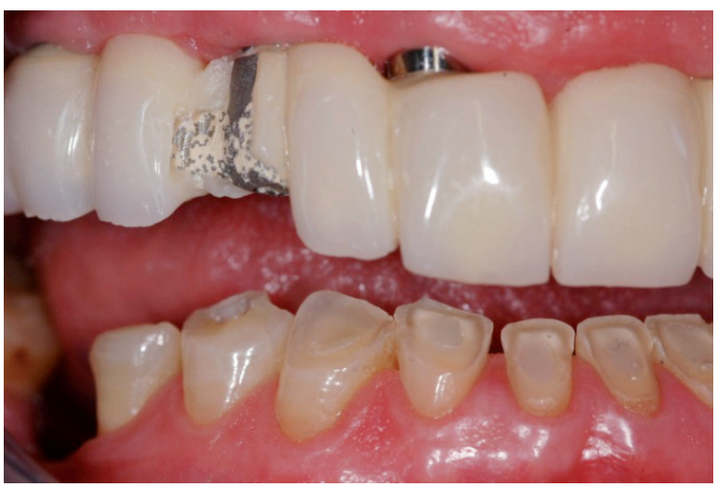

Figure 5.
Increased tooth wear of mandibular teeth cause by a combination of attrition and erosion.

to acid erosion. Sometimes, it could be difficult to distinguish if teeth with missing enamel and dental surface breaks are affected by hypoplasia, that is a quantitative reduction of enamel formation or by acids consumption. Erosion mediated by acids may also be undistinguished from enamel hypomaturation, when diffused opacities are observed. Then, the area of the opacities or structure deficiencies must be carefully observed and all the mouth have to be analyzed to understand if those defects are localized in only a part of the mouth or widespread. A correct anamnesis of the patient must also be performed regarding diet habits, gastrointestinal pathologies or drugs assumption. Dietary analysis and advice regarding erosion and sugars are fundamental to reduce further problems in teeth affected by amelogenesis imperfecta [77]. Conversely, children with AI and DI will often avoid ice cream and fridge-cold products because of the hypersensitivity and this constitute a protective factor. However, a lot of other cariogenic or acidic products may be responsible for erosion processes.

In the advanced state, the erosion can extend into dentin. The level of painful hypersensitivities as well as the esthetic or functional limitations are generally related to the extension of the erosion, although sometimes an individual component for dentin hypersensitivity may exacerbate this phenomenon. Also in this case, poorly mineralized dentine, and tubular defects in dentinogenesis imperfecta may express as extensive tooth wear, similarly to advanced case of erosion with similar hypersensitivity.

From an etiologic point of view, erosive defects can be distinguished in endogenous and exogenous. The consumption of acidic food and drugs, as well as occupational acid exposure such as wine tasters and professional swimmers, are considered extrinsic exposures [78]. Instead, intrinsic erosion is intended when gastric fluids come into contact with the oral cavity, especially in patients suffering of gastrointestinal reflux disease, eating disorders, and/or alcohol abuse [79].

Usually, a palatal and occlusal localization of the erosion defects is due to an intrinsic erosion, while an extrinsic erosion affects the labial surfaces of the anterior teeth [80]. Both types of erosion produce deleterious effects on dental elements, with a pattern of destruction dependent on the erosivity of the erosion-causing solution (pH, buffer capacity, and mineral concentration), and also on the frequency and type of acid exposure. However, as gastric fluid is evaluated as 1 in the pH scale and is provided with a high amount of free acid, its erosive potential is higher than that of extrinsic acids [81]. Moreover, patients with eating disorders often show xerostomia because of the lower salivary flow rate caused by the general dehydration or by the antidepressant drugs, which could further increase the risk of developing erosive lesions.

3.4 Abfraction

Abfraction or Non Carious Cervical Lesions (NCCL) have been used to describe wedge shaped cervical lesions as a wear defect [82]. It is recognized as the loss of cervical tooth tissues induced by mechanical loading which led to enamel and dentin flexure and failure [83]. Some biomechanical analyses show that the most important area of stress concentration is located at the cervical areas of the teeth in response to overloading, that leads to initiation of a cervical lesion [84, 85].

Another study, using FEM, suggested that oblique loading on the tooth stretches the enamel surface near the cemento-enamel junction causing plastic deformation at the cervical area [86]. It was seen that lateral forces produce compressive stresses on the side toward which the tooth bends and the tensile stresses are on the other side. These stresses create microfractures at the cervical region which propagate perpendicularly to the long axis of the tooth, leading to a localized defect around the CEJ [87]. The tensile forces could disrupt the hydroxyapatite (HA) crystals of the enamel structure, allowing saliva and other small molecules to penetrate between the prisms and prevent re-establishment of the interprismatic bonds on release of the stress (**Figure 6**). Ultimately, when the enamel breaks away at the cervical margin and exposes the dentin, the process continues in this way and may accelerate because of the structure of the dentin [82].

Cervical lesions depend on type and severity of the etiologic factor, and not all these lesions require restorations. They appear primarily at the cervical region of the dentition and are typically wedge-shaped, with sharp internal and external line angles [55].

Treatment planning of non carious cervical lesions is based on the reduction of stress concentration in order to strengthen the tooth, the prevention of dentin hypersensitivity with major pulp protection and the modification of oral hygiene habits, improving also the esthetics [82]. Composites and glass ionomer restorations can be adopted if lesions are not too much extended. On the other hand, metal crowns can be used where the masticatory load is higher. In order to treat hypersensitivity, dentin bonding agents, fluoride varnishes and other desensitizing agents may be useful. Gnatologic devices also can be fabricated to protect teeth during night, however changing of dietary and oral habits is mandatory [88, 89].

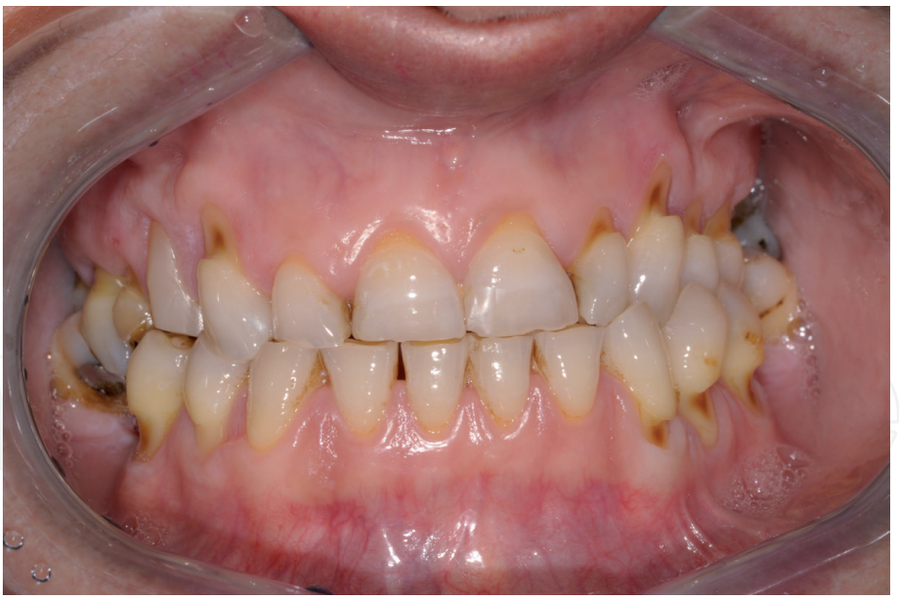

Figure 6.
Abfraction lesions associated with moderate tooth wear.

4. Diagnosis and management of patients with developmental dental defects and tooth wear

Tooth wear is multifactorial in origin [51]. The major factors responsible for tooth wear should be identified starting from a correct and thorough anamnesis of the patient in order to establish a predictable treatment plan. Several signs may result useful in the differential diagnosis process and the appearance of worn tooth surfaces resulting from the various types of wear differ. In order to make a correct diagnosis of the etiology of tooth wear it is fundamental not only to observe the wear pattern but also to investigate if any erosive or abrasive factor is present in the anamnesis. However, if a clear etiological factor is not find, the observed tooth wear may be due to the mechanical type. However, identification and recognition of developmental dental defects is of extreme importance (**Table 1**) [23, 94]. In fact, early diagnosis and preventive care are essential for the successful treatment of developmental dental defects. Children with a family history of amelogenesis or dentinogenesis imperfecta, or medical syndromes commonly associated with them such as prematurity of birth or hypophosphatemia should be assessed for developmental dental defects as soon as the teeth erupt. Defects in primary teeth may possibly indicate a risk also for permanent dentition [7].

For children with developmental dental defects, a preventive program should be instituted immediately after diagnosis. Neutral sodium fluoride gels or varnishes professional applications every 3/6 months, in addition with calcium and phosphate rich agents (casein phosphopeptide-amorphous calcium phosphate, CPP-ACP) are recommended to reduce caries risk and developmental opacities in teeth with enamel hypoplasia [95]. Because of the structural weakness of the teeth with developmental dental defects, other important recommendations are the same as erosive protection advices such as reduced consumption of acidic food, diet and soft drinks, control of eventual psychosomatic disorders, because of the possibility of frequent vomiting [90]. It is also important to consider that the risk of erosive lesions is increased when acid or soft drinks are assumed by children from a feeding bottle at bed- or nap-time, because of the lower salivary flow rate during sleep [96]. Furthermore, several drinking habits (sips drinking, use of a straw in direct contact with teeth, and intensive rinsing) cause a prolonged pH drop in the oral

	Etiology	**Clinical signs**	**Preventive and possible therapeutic options**
Attrition	Friction between opposing teeth [72]	Occlusal tooth wear	Teeth prosthetic or conservative restorations [1, 2]
Erosion	Contact between acid substances and teeth [43]	Vestibular, palatal and/or occlusal tooth substance loss	Avoid acid foods and drinks consumption [90, 91]
Abrasion	Friction between teeth and foreign body [72]	Cervical vestibular tooth substance loss	Avoid horizontal toothbrushing technique and dental abrasive habits [48, 52]
Enamel hypoplasia	Quantitative reduction of enamel formation [12]	Thin enamel area with surface pitting or vertical grooving on several teeth	Microabrasion and restorative or prosthetic treatment [12, 92]
Enamel hypomineralization	Reduced enamel mineralization [40, 93]	Soft and/or discolored enamel	Fluoride applications, restorative and/or prosthetic treatment [16, 17]
Dentinogenesis imperfecta	Alterations in collagen proteins [47]	Tooth discoloration, enamel fractures, pulp obliteration	Prophylactic coverage with stainless steel crowns, Fluoride applications [7]

Table 1.
Summary table of the etiology, clinical signs and preventive and therapeutic options of developmental dental defects and tooth wear conditions. Clinicians must consider possible associations between these two pathological entities.

cavity compared to a short consumption [97]. Then, patients should restrict the consumption of acidic food and drinks only to main meals. Acidic beverages should be consumed cool and as fast as possible in order to reduce their erosivity. Some foods as yogurts that have high concentrations of calcium and phosphate, result non-erosive despite their low pH [91].

When tooth wear is already present, the treatment planning in children with extensive enamel defects due to may involve complex restorations, orthodontics, exodontia and prosthodontics [77].

Normally, without any developmental dental defects, the treatment planning depends on the severity of tooth wear. The amount of tooth wear necessary for intervention is not clear from the scientific literature, even if with the Smith and Knight index [98], the threshold to start restorative treatment is set once dentine was involved. A recent paper summarizes when it is recommended a restorative treatment [99]. Another paper indicates several techniques and treatment strategies for tooth wear, clearly distinguishing between pathological and physiological tooth wear; it is also highlighted that dentist has to detect the speed and severity of tooth wear process in order to decide when intervening [100]. However, difficulties in detecting a pathological dental loss at early stages differently from physiological loss, is challenging for the dentist. A complicating factor is also that tooth wear may be cyclical and can be inactive in the majority of the patients, despite obvious wear facets in their dentitions [101].

However, in developmental dental defects, because the structural weakness of the hard tissues leads to its readily deterioration under masticatory stresses

and both amelogenesis and dentinogenesis imperfecta are associated with rapid toothwear and crown fractures, protection from toothwear is recommended soon after eruption [102]. Ideally, restorative stabilization of the dentition should be completed before excessive wear and loss of vertical dimension occur [103]. Guidelines for the treatment of developmental dental anomalies have been established by AAPD (American Academy of Pediatric Dentistry) [104]. For developmental enamel defects, treatment should begin as soon as possible according to patient compliance in office dental care. Early identification and preventive interventions are critical for infants and children with enamel defects due to amelogenesis imperfecta in order to avoid the negative social and functional consequences of the disorder. The appearance, quality, and amount of affected enamel and dentin will dictate the type of restorations necessary to achieve esthetic, masticatory, and functional health. Depending on the severity of enamel defects and tooth wear, treatment can range from bleaching and/or microabrasion [92] to composite resin, porcelain veneers [105] or full coverage restorations with crowns placement [39].

Treatment of dentinogenesis imperfecta frequently includes preventing severe attrition associated with enamel loss and rapid wear of the poorly mineralized dentin, rehabilitating dentitions that have undergone severe wear, optimizing esthetics, and preventing caries and periodontal disease [104].

Stainless steel crowns are a highly durable restoration choice for the prophylactic coverage of teeth affect by developmental dental defects. In teeth with dentine defects, they reduce the risk of pulp exposure and infection, especially in some types of dentine defects (hypophosphatemia) [28]. Fluoride applications and desensitizing agents may also diminish tooth sensitivity [106]. In teeth affected by enamel hypoplasia both primary and permanent molars show a reduction in tooth sensitivity and in cusp fractures after prosthetic rehabilitation with stainless teel crowns. This also helps to maintain space and crown height, important also for orthodontic issues. The crowns are best inserted using a conservative technique, originally proposed by Seow, that involves a minimal removal of tooth structure in order to protect teeth with large pulps and dentin defects [28, 107]. In adulthood, the stainless steel crowns may be replaced with gold or porcelain crowns to provide long term protection of the teeth.

It should also be considered that marginal leakage around restorations and recurrent caries with eventual pulp involvement may be determined from the enamel deterioration [108, 109]. Materials as resin modified glass-ionomer cements and polyacid modified composites should be used for restoring teeth affected by enamel defects in order to take advantage of the optimal bonding of these material with both dentine and enamel [110]. However, despite their esthetic value, composite resins have low adhesion to poorly mineralized enamel. Then, it is important to consider the amount of tooth wear in order to proceed with conservative or prosthetic rehabilitation.

In cases with significant loss of vertical dimension because of tooth wear, the reestablishment of a more normal vertical dimension is crucial for a correct function, mastication and esthetics. Cases showing severe loss of coronal tooth structure and vertical dimension have to be considered candidates for overdenture therapy. Overlay dentures placed on teeth that are covered with fluoride-releasing glass ionomer cement have also been used with success [111].

Conflict of interest

The authors declare no conflict of interest.

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